than 20 μmoles/liter, the derived isotopic and direct enzymatic measurements of plasma oxalate were carried out. Where the isotopic and the enzymatic methods could be applied simultaneously, the correlation was good. Raised plasma oxalate and raised exchangeable oxalate pool were attributable to both primary hyperoxaluria and chronic renal failure and were seen also in the 3 stone-formers, who were assumed to be intestinal hyperabsorbers of oxalic acid, but for any given creatinine clearance the plasma oxalate and exchangeable oxalate pool were set considerably higher with primary hyperoxaluria than they were with the other conditions. In most normal subjects and most patients, the ratio of clearance of oxalate to clearance of creatinine was greater than 1.

Effect of prostacyclin on platelets in hemodialysis: Dialysis without anticoagulation. *H. F. Woods, M. J. Weston, and S. Bunting. Renal Unit, King's College Hospital, London, and Wellcome Research Laboratories, Beckenham, Kent, England.* Prostacyclin, produced by vascular endothelium, is the most potent inhibitor of platelet function known to man. To assess its effects on platelet-foreign-surface interactions during hemodialysis, 10 greyhounds were dialyzed with Cuprophan coils (Travenol) and standard dialysis equipment for 90 min. At the end of dialysis, arterial platelet counts (% of initial) in 5 dogs in which prostacyclin had been infused (115.7 ± 8.6) were significantly higher than in 5 animals in which heparin alone was given (77.8 ± 16.8) ($P < 0.05$). Prostacyclin reduced the extraction of platelets by the dialyzer. The screen filtration pressure of blood leaving the dialyzer, a measurement of platelet aggregates and microemboli, was not elevated in the animals which received prostacyclin (80 ± 11.3 mm Hg) but rose significantly in those which received heparin only (249 ± 57 mm Hg) ($P < 0.02$). In another 5 greyhounds infused with prostacyclin, but which received no heparin, dialysis was completed without significant clotting within the dialyzer or lines. The platelet count did not fall, the screen filtration pressure did not rise, and tests of blood clotting were not altered compared with predialysis values. Thus prostacyclin prevents platelet activation during hemodialysis and enables dialysis to be carried out without anticoagulation.

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Angiography of arteriovenous hemodialysis fistulas. *P. Aubert, A. Bernard, J. Bachet, P. Quentric, B. Goudot, and J. Guédon. C.M.C. Foch 40, Suresnes, France.* During the past 5 years, 60 angiographies of hemodialysis arteriovenous fistulas (AAF) were performed on 32 patients. Three methods were used: retrograde catheterization through femoral artery (3 cases), direct arterial cannulation (6 cases), and retrograde venography (51 cases). Indications for AAF included insufficient blood flow during dialysis or poor development of the fistula (30 cases), abnormal return pressure of the blood during hemodialysis (16 cases), and others (14 cases). Roentgenographic abnormalities were found in 86.7% of AAF, which was correlated well with clinical findings in 73%. Reliable diagnosis was impossible in 8.3% of AAF, mainly because of inadequate methods, and 5% appeared normal. Roentgenographic findings identified vascular stenosis (56%) or thrombosis (16%), aneurysms (19%), and miscellaneous (9%). AAF was useful for planning appropriate medical or surgical treatment in 58% of the studies; in 42%, radiologic abnormalities were thought to be compatible with prolonged survival of the fistula, and no treatment was performed. Retrograde venography seems the method of choice for AAF because it is safe and effective in most cases.

Urinary lactic deshydrogenase isoenzyme 5 (LDH5) and urinary tract infection in children. *F. Bouissou, P. Barthe, J. P. Thouvenot, P. Bourely. Service de Médecine Infantile C, Laboratoire de Biochimie II, Hôpital Purpan, Cedex, France.* It has been stated that an increase in urinary excretion of LDH5 is helpful in topographic diagnosis of urinary tract infection (UTI). Sixty-three children (mean age, 6 years) with significant UTI were studied. They had a significantly higher ($P < 0.01$) LDH5 excretion than control subjects free of UTI had (65 normal children, 32 with renal failure, 29 with the nephrotic syndrome, 88 with uropathy). The presence of renal failure did not influence the results. A good correlation was found with inflammatory tests, quantitative bacteriologic count, and leucocyturia. No correlation was found with immunologic parameters (antibody-coated bacteria in urine, and serum antibodies). According to the results of the preceding tests and to the presence or not of malformative uropathy, a quantitative score was attributed to each patient. We found a significantly ($P < 0.02$) higher urinary elimination of LDH5 in patients with high scores (renal infection) than in those with low scores (bladder infection). We conclude that high urinary LDH5 level does not reflect only renal parenchymal infection. LDH5 excretion may increase with leucocyturia. It is only a presumptive test, among other techniques, for topographic diagnosis of UTI. These results conflict with previous data obtained in adult patients.

Theophylline influence on the ferritin pathway through the glomerular basement membrane in the rat. *J. Cambar and P. Gendre. Department of Physiology, U.E.R. of Pharmaceutical Sciences, Bordeaux, France.* The present study compares the ferritin particle density (particle number/cm² of micrographic area) within the capillary lumen and the glomerular basement membrane (GBM) in rats infused either with isotonic saline solution (0.15 ml/min flow) (control) or with a 3 mg/ml theophylline solution (treated). Particle density within the capillary lumen was 25.27 ± 3.93 part./cm² in control rats and 7.7 ± 2.87 after theophylline infusion (− 228%). Within the GBM, particle density was 4.68 ± 1.85 part./cm² in the control rats, whereas it was 17.11 ± 3.9 in the treated animals (+ 265%). Ferritin density increase within the GBM indicates an augmentation in the proteic tracer passage through the glomerular barrier. This interpretation is confirmed by the parallel decrease in particle density within the capillary lumen. Many data, which we have recently described, contribute to explaining the present observations. Indeed, we have demonstrated that theophylline, as a cyclic AMP phosphodiesterase inhibitor, increases afferent arteriole diameter and isolated glomeruli diameter, probably by myorelaxant effect on smooth muscle fibers, thus increasing the cortical blood flow. Morphometric ultrastructural studies have confirmed diam-

eter increase of endothelial pores and epithelial slits. Such structural modifications of the glomerular filter after theophylline injection may increase the transglomerular flow of water, solutes, and macromolecules. Indeed, we have noted increased diuresis (+ 36%), GFR (+ 133%), and urinary protein excretion (+ 73%), which is in agreement with the present increase in transglomerular tracer passage.

Action of a natriuretic factor on the isolated perfused rat kidney: Absence of relationship between this factor and prostaglandins. *P. Cambier and J. P. Godon. Institute of Medicine, State University of Liège, Belgium.* In recent works, we have demonstrated that the presence of a natriuretic factor of renal origin with a molecular weight of 45,000 daltons was necessary to allow an adequate natriuresis by the kidney after an acute or chronic salt load. Its presence has always been evidenced by a bioassay on anesthetized rats. The two following questions were thus raised: *(1)* Does this factor depend on an enzyme-substrate system? *(2)* Is there a relationship with renal prostaglandins? The natriuretic factor, when injected intraarterially or added to the cell-free solution perfusing isolated rat kidneys, induces a very high natriuresis (fractional excretion expressed by the mean differences from initial values: +6.01 ± 2.14%) as compared to the infusion, in identical conditions, of the extract solvents, − 0.98 ± 2.31%. Furthermore, if the rat kidney donors are prepared by indomethacine, the injection of the natriuretic factor induces a natriuresis quite similar to that observed in unprepared kidneys. This natriuresis is stimulated without modification of glucose transport. In conclusion, the natriuretic factor acts directly on an isolated cell-free perfused rat kidney and thus does not depend on an enzyme-substrate system of extrarenal origin (comparable to the renin-angiotensin system). Moreover, the inhibition of prostaglandin synthetases does not modify the renal response to the natriuretic factor: the presence of renal prostaglandins is not necessary for its action.

Renal lesions in a Rothmund-Thomson disease. *J. M. Chantraine, D. Brumioul, C. Dechenne. Department of Pediatry and Department of Pathology, Liège University, Belgium.* A Rothmund-Thomson disease was diagnosed on the following symptoms: poikilodermia, dwarfness, and a rapidly progressive bilateral cataract in a 6-year-old girl. Moreover, a severe, persistently high blood pressure (170/120 mm Hg) without any urinary abnormality was observed. This symptom had never before been described in this syndrome. A kidney biopsy studied by optical microscopy revealed calcifications involved in hyaline hypersecretion in the intima of some middle size arteries; arterioles appeared normal, glomeruli showed a small increase of the mesangial matrix. With electronic microscopy, an important hypersecretion of membranous material was observed in the intima of most of the arterioles, and, to a lesser extent in the mesangial area; glomerular capillary basement membranes were thick for a child of that age (3.500 to 5.500 Å). Cells and tubular basement membranes were normal. Notwithstanding the possible role of the relative ischemia in glomerular basement membrane and mesangial thickening, this observation suggests that, in this case, the disease could be the result of a collagen hypersecretion of type 3 and 4 by the endocapillary cells, i.e., endothelial and mesangial cells. Moreover, the appearance of a cataract and of calcic deposits in arteries could be the result of local biophysical abnormalities of these collagens.

Basement membrane (BM) abnormalities in three congenital renal diseases. *C. Dechenne, J. C. Davin, J. M. Chantraine. Anatomo-pathological Department, Pediatric Department, Liège University, Belgium. (1)* A 10-week-old boy with a congenital nephrotic syndrome classified as type III according to the criteria of R. Habib, underwent a renal biopsy. Using electron microscopic (EM) studies, we observed: *(a)* focally, a plurilamellar aspect of the glomerular BM, *(b)* a very thin GBM for the age of the baby with many "clear" subendothelial deposits, *(c)* local accumulations of BM material in the glomerular as well as in the capsular BM, suggesting the beginning of hyalinosis. *(2)* A 5-year-old daughter of a recently transplanted deaf man underwent a kidney biopsy for intermittent microscopic hematuria. The clinical data excluded a poststreptococcal or a Berger's disease. EM studies revealed an inhomogeneity of the GBM with focal inclusions of dense granules, morphologic abnormalities observed in Alport's syndrome. We assume that these lesions have the same origin. *(3)* A 19-year-old boy underwent a kidney biopsy for intermittent microscopic hematuria also known in the father, without any functional alteration. By EM a very thin GBM (≤ 2.000 Å) with focally a BM thinner than 1.000 Å was observed. These observations suggest that the morphologic changes are in correlation with the urinary abnormalities, but the point of their origin(s) or their mechanism(s) is not resolved.

Treatment of chronic renal failure by hemofiltration with regeneration of ultrafiltrate on sorbents before reinjection. *P. Dequiedt, F. Goudaert, G. Lelievre, and A. Tacquet. Hôpital A. Calmette, Lille, France.* We compare the results of two hemofiltration procedures in the same 56-year-old patient: *(1)* hemofiltration alone without adsorption, for 6 months and, *(2)* hemofiltration with adsorption of the ultrafiltrate on coconut-activated charcoal before reinjection for an additional 6 months. Treatment was performed for 4.5 hours three times a week. Preparation of the sorbent column and clinical application in the human are described. The two techniques were well tolerated, and their efficiency was very satisfactory, particularly on acid-base balance. Although the sorbent we used removed a low amount of urea, after 4 months of hemofiltration-adsorption-reinjection, azotemia did not rise. This fact supposes a metabolic adaptation phenomenon.

Hypertension in chronic hemodialyzed patients: Hemodynamic study before and after water and salt depletion by hemodialysis. *G. Dongradi, J. P. Fendler, J. C. Kahn, P. Rocha, A. Ferreira, and D. Hillion. Department of Internal Medicine and Nephrology, Poissy C.H.I.* In 15 chronic hemodialyzed patients (CHP) free of patent heart disease, hemodynamic study was performed during bed rest and during intense sitting exercise before and after water and salt depletion. Cardiac performance in 7 hypertensive patients (HTP) was compared, before and after dialysis, with that in 8 normotensive patients (NTP). At bed rest, systemic arterial pressures, systolic, diastolic, and mean pressures were correlated with pulmonary wedge pressure (PWP) before dialysis ($P < 0.01$) and after dialysis ($P < 0.001$), but were not correlated with right atrial pressure (RAP) either before or after dialysis; PWP was higher in the HTP than in the NTP before and after dialysis; there was no difference in RAP between NTP and HTP before and after dialysis: total blood volume did not appear to be greater in the hypertensive patients than in the normotensive patients. During intense exercise, cardiac index values were the same in the HTP before dialysis (9.4 ± 2.4 liters/min/m²) and after dialysis (9.2 ± 1.8 liters/min/m²) as in the NTP before dialysis (9.4 ± 1.8 liters/min/m²) and after dialysis (9.0 ± 1.6 liters/min/m²); heart rate rose in the HTP before and after dialysis to a level slightly less than that found in the NTP: there was no evidence of left ventricular failure in the hypertensive patients as compared to the normotensive patients. Since before and after dialysis total blood volume and left ventricular performance were no different in hypertensive patients and in normotensive patients, the higher PWP value in hypertensive patients as compared to normotensive patients could be hypothetically related to a lower left ventricular compliance and mainly to a lower systemic venous capacitance responsible for a higher cardiopulmonary blood volume. If this latter hypothesis is confirmed, hypertensive patients as compared to normotensive patients could have a more marked sympathetic activity.

Vasopressin (VP) -induced changed in the epithelial surface of toad urinary bladder. *M. Dratwa, A. LeFurgey, and C. C. Tisher. Département de Néphrologie, Hôpital Universitaire Brug-*

mann, Brussels, Belgium, and Duke University, Durham, North Carolina, USA. Previous scanning electron microscopy (SEM) studies have revealed changes in the surface architecture of granular (GR) cells of toad urinary bladder stimulated by VP. However, there is still controversy with regard to the effects of VP per se as separate from changes in cell volume or shape due to VP-induced osmotic water flow. The present study was initiated to clarify this issue. Hemibladders were stimulated with VP (20 mU/ml) in the absence of transepithelial osmotic gradient so that no osmotic water flow could be elicited. When examined with SEM, the luminal surface of GR cells from paired control preparations not exposed to VP consisted of arborizing ridges. In striking contrast, GR cells of VP-stimulated tissue exhibited closely packed, but discrete microvilli. No cell swelling occurred. Similar modifications were seen in bladders challenged with cAMP. On the other hand, an increase in cell volume induced by incubation of the tissue in a hypotonic solution did not result in microvillus formation, nor did a change in cell shape caused by bladder contraction after exposure to acetylcholine. These findings establish that surface changes in GR cells of toad bladder do occur under the influence of VP per se via its intracellular mediatior cAMP since they can be observed in the absence of osmotic gradient but not after a geometric deformation comparable to that induced by osmotic water flow.

Absence of increased motor nerve conduction velocity after parathyroidectomy in dialysis patients. *T. Drüeke, N. Chkoff, S. Di Giulio, J. Zingraff, S. Delons, N. K. Man, P. Jungers, and J. Crosnier. Department of Nephrology and INSERM U 25, Necker Hospital, Paris, France.* Excess parathyroid hormone (PTH) has been recently incriminated in the etiology of uremic polyneuropathy, and an inverse relation has been found between serum PTH concentration and motor nerve conduction velocity (MNCV). We have analyzed in a retrospective study whether the dramatic decrease in circulating PTH after parathyroidectomy (PTX) was followed by an increase in MNCV. Twelve uremic patients on intermittent hemodialysis were selected in whom sufficient biochemical and nerve conduction data was available for analysis. They had undergone subtotal PTX between 1974 and 1976 because of uncontrolled, severe secondary hyperparathyroidism and had repeated MNCV determinations. Their mean ± SEM plasma PTH concentration was 124 ± 19 and 29 ± 8 ng of protein/ml before and after PTX, respectively ($P < 0.01$) (normal of the laboratory, 4 to 8 ng of protein/ml). Their mean plasma calcium concentration significantly decreased to 7.7 ± 0.4 mg/dl immediately after PTX as compared to 10.1 ± 0.2 mg/dl before surgery ($P < 0.001$). None of the patients had clinical manifestations of motor neuropathy. MNCV which was measured every 3 to 6 months in all of them was 39.2 ± 1.6 m/sec before PTX (mean of 2 to 3 values in each patient) and 40.3 ± 1.3 m/sec during 9 to 15 months following PTX. This change was not significant. Moreover, no inverse relation was found between plasma PTH concentration and MNCV in this group of patients. It is concluded that correction of PTH excess in uremic patients did not lead to a substantial improvement in their motor nerve conduction velocity.

Different ability to produce antibodies to heterologous tubular and glomerular basement membranes in two strains of mice. *D. Erard, D. Moulonguet-Doleris, M. T. Auffrédou, P. Mahieu, and P. Galanaud. INSERM U 131, Clamart, France.* Glomerular basement membrane (GBM) and tubular basement membrane (TBM), purified from dog kidney, were used as antigen in complete Freund adjuvant (CFA). Balb/c mice received 8 weekly injections of GBM, TBM, or CFA alone. Serum collection and kidney biopsies were performed every 2 weeks, and 1 week after the last injection. The circulating anti-GBM and/or anti-TBM antibodies were titrated with radiolabeled soluble GBM or TBM antigen, in a modified Farr assay. Antibodies were detected in the sera as early as week 5 and exhibited a specificity for the antigen used. Linear IgG deposits were observed after the eighth injection. The eluted IgG could be fixed on normal kidney sections and transferred to normal Balb/c mice. Mice immunized with GBM exhibited strong linear deposits on both GBM and TBM, whereas mice immunized with TBM exhibited linear deposits mainly on TBM. In contrast, when the same immunization was performed in C3H mice, no consistent deposits were observed on TBM, whether the antigen used was TBM or GBM.

Acute renal insufficiency following intoxication by glafenin. *M. Estenne, R. Naeije, P. Ketelbant-Balasse, P. Gausset, and J. C. Demanet. Service de Médecine, Hôpital St. Pierre, Bruxelles, France.* A 35-year-old man, after ingestion of 3 g of glafenin with the purpose of committing suicide, was admitted complaining of lumbar pain and oliguria. Immediate and subsequent physical examinations revealed no abnormalities. Blood urea was 88 mg/100 ml, creatinine was 7.4 mg/100 ml, urinary sodium excretion was 11 mEq/24 hr, and there was no proteinuria. No oliguria was observed. No circulating immune complexes were found. A lymphocyte transformation test to glafenin was negative. After reaching maximum levels of 121 and 12 mg/100 ml, respectively, at day 6 after intoxication, blood urea and creatinine progressively returned to normal. A renal biopsy was performed on day 7 after intoxication. Light microscopy showed discrete glomerular and tubular lesions. Electron microscopy showed that at the glomerulus, there was a partial epithelial-cell foot-process fusion and lysosomal granular deposits in the epithelial cells, and that at the proximal tubule, there were lysosomal granular deposits. Immunofluorescence microscopy showed abundant interstitial fibrinogen and immunoglobulin G deposits. Acute tubular necrosis lesions have been described in most of the reported cases of acute renal insufficiency following glafenin ingestion. Our study suggests that glafenin would also be toxic at the glomerular level, which has not been reported yet.

Tubular transport mechanisms of aminoglycosides. *J. Fabre, R. de Sousa, and M. Rudhardt. Departments of Medicine and of Physiology, Geneva, Switzerland.* It is generally assumed that aminoglycosides accumulate in the cells of the proximal convoluted tubule through reabsorption at their luminal pole. However, the mechanisms involved seem to be more complex. *(1)* Wistar rats whose ureters had been ligated 20 hours previously received 4 mg/kg gentamicin or 25 mg/kg amikacin i.p. As compared to normal rats, their levels of antibiotic 6 hours after injection were higher in the cortex, which largely consists of proximal convoluted tubules (bioassay; means of 6 rats ± SD):

	Control group	*Ligated ureters*	
Gentamicin, μg/g	123 ± 20	310 ± 71	$P < 0.001$
Amikacin, μg/g	156 ± 21	729 ± 117	$P < 0.001$

Thus, in spite of an important decrease in glomerular filtration and consequently in reabsorption, aminoglycosides still penetrate the tubular cells in larger quantities, probably by the way of their basal pole. *(2)* The adjunction of an organic base (quinine, 35 mg/kg) paradoxically increased the accumulation of gentamicin in the cortex of normal rats (means of 6 rats ± SD; $P < 0.05$ at 1, 6 and 72 hours, $P < 0.001$ at 24 hours).

	After injection			
	1 hr	*6 hr*	*24 hr*	*72 hr*
Gentamicin, 4 mg/kg	81 ± 10	123 ± 20	96 ± 21	71 + 9 μg/g
Gentamicin + quinine	129 ± 17	189 ± 37	168 ± 32	116 ± 39 μg/g

This interaction would seem to indicate that even under normal conditions gentamicin follows the pathway of secretion of organic bases but, in the presence of quinine, the competition takes place apparently at the luminal pole to leave the cells of the convoluted proximal tubule. *Summary.* Aminoglycosides are subject to bidirectional tubular transport: they can both be reabsorbed and penetrate the cells of the proximal convoluted tubule at their basal pole, presumably through the pathway of secretion of organic bases.

Glomerulonephritis induced in mouse by *Eschericia coli*. *G. J. Fournié, M. A. Mignon-Conte, M. Gayral-Ta Minh, J. J. Conté, Laboratoire d'Immunopathologie Rénale. Service de Néphrologie et d'Hémodialyse. C.H.U. Toulouse-Purpan, Toulouse, France.* To analyze the mechanisms by which infectious agents may induce glomerulonephritis, we infected mice with *Escherichia coli* (E. coli) and kidney lesions were evaluated by optical and electron microscopy and by immunochemical procedures. It was found that as early as a week after the injection, heavy deposits of IgG and IgM were present in the mesangium and along glomerular capillary walls. Histologic studies revealed endocapillary proliferation of cells and dense deposits in mesangial and subendothelial areas. The mechanism of these lesions was investigated. In serum, large amounts of DNA were present in all mice 1 day after injection. This DNA disappeared on the following day. Anti-DNA antibodies were detectable on day 3 and reached a peak on days 7 to 14. Thirty-five days later, significant levels of anti-DNA antibodies still remained in circulating blood. C1q binding material was also found after 1 week, suggesting the presence of circulating immune complexes. Anti-E. coli antibodies appeared generally on day 7. All these findings mimic the data observed in mice infected by bacterial lipopolysaccharide (LPS) or lipid A. However, the lesions are much more severe in mice infected by E. coli than in mice injected with LPS, and this model seems closer to the human pathological situation than the LPS model does. Further studies include the investigations on the nature of immune complexes deposited in kidney and on the role of the formation of DNA-anti-DNA immune complexes on glomerular and tubular basement membrane in the development of renal lesions.

Moya-Moya syndrome and renal artery stenosis. *M. Godin, A. Helias, M. Tadie, J. P. Fillastre, and P. Creissard. Centre Hospitalo-Universitaire de Rouen, France.* A 30-year-old female presented with right and left recurrent hemiparesia without sequela. Followup disclosed extensive progressive narrowing of the carotid arteries after the carotid siphon and abnormal capillary vasculature at the base of the brain. This image is typical of the Moya-Moya syndrome, which was initially observed only in Japanese subjects. Vascular manifestations of the syndrome are signs of cerebral circulatory insufficiency, or hemorrhage in young subjects. Wider use of cerebral angiography has led to easier identification, and the syndrome no longer appears geographically limited (20 observations reported in France). The etiology of the syndrome is unknown. Laboratory investigations are fragmentary. In studying, in our case, the widest possible accumulation of data, it was impossible to demonstrate either an immunological, inflammatory mechanism or a metabolic or platelet abnormality. Hypertension is rare in the Moya-Moya syndrome. However, in this case we observed arterial pressure of 260/120 mm Hg, associated with a right hypogastric murmur. Extensive stenosis of the right renal artery was observed with angiography, as were other arterial abnormalities (internal and external ilia, mesentery, left kidney). A review of the literature disclosed other associated renal malformations such as polycystic disease and arterial lesion nephropathy. Few pathologic and anatomic studies have been published, but two authors mention intimal hyperplasia outside of the cerebral area (heart, liver, kidney, spleen). These findings raise the question whether the vascular involvement in the Moya-Moya syndrome is limited. When hypertension is associated, the possibility of renal vascular abnormalities should be considered.

Hyperphosphatemic acute renal failure (ARF) following therapy of lymphosarcoma. *A. Kanfer, J. Roland, F. Chatelet, G. Richet. Services de Néphrologie et d'Anatomie pathologique, Hopital Tenon, Paris, France.* In a 43-year-old man hyperphosphatemia with ARF occurred during chemotherapy of relapsing lymphosarcoma (poorly differentiated type). Relapse was indicated by disseminated enlarged lymph nodes and bone marrow infiltration by lymphoblasts. On day 0, the patient received i.v. cyclophosphamide (1.2 g), doxorubicine (100 mg), vincristine (1.5 mg), prednisolone (120 mg), and urate oxydase. Two days later (day 2) lymphadenopathy had disappeared. Serum phosphorus rose from 4.3 mg/dl (2 days before chemotherapy) to 24.5 mg/dl (day 3). Over the same period, serum creatinine rose from 1.2 to 3.0 mg/dl, whereas serum calcium fell from 10.0 to 5.7 mg/dl and serum uric acid from 13.2 to 5.5 mg/dl. Ureteral obstruction was excluded. The patient became anuric and had to be dialyzed on 5 occasions. On day 9 serum phosphorus was 9.5 mg/dl, and serum calcium was 9 mg/dl. Twelve days after the onset of ARF, the patient died of gastrointestinal bleeding. Renal necropsic findings disclosed diffuse flattening of tubular epithelium with marked tubular dilatation and presence of calcium crystals in several tubular lumina; there was no neoplastic parenchymatous infiltration. This extreme hyperphosphatemia most probably *(1)* originated from chemotherapy-induced brisk lysis of lymphoblasts with a high phosphorus content, *(2)* provoked hypocalcemia and ARF with nephrocalcinosis, intrarenal precipitation of calcium being triggered by a very high calcium phosphorus product (139.65). *Conclusion.* Acute postchemotherapy hyperphosphatemia is a possible cause of oligoanuria in hematologic malignancies.

Antiglomerular basement membrane (GBM) antibody-induced glomerulonephritis after solvent exposure. *D. Kleinknecht, L. Morel-Maroger, P. Callard, J. P. Adhémar, and P. Mahieu. Service de Néphrologie, Hôpital de Montreuil, Unité de l'INSERM U 64, Hôpital Tenon, Paris, Laboratoire d'Anatomie Pathologique, Hôpital de Bondy, France, and Hôpital de Bavière, Liège, Belgique.* Rapidly progressive glomerulonephritis with definitive anuria was observed in two young women aged 22 and 17 after solvent exposure. In patient 1, anuria occurred 3 years after a Hodgkin's disease treated by chemotherapy and irradiation (12,000 rads). No antecedent was found in patient 2. In both, renal biopsy showed diffuse epithelial crescents in all glomeruli with linear deposits of IgG along the GBM. Circulating anti-GBM antibodies were initially present in serum in a high titer, either by indirect immunofluorescence (IF) or by radioimmunoassay (34% and 28%), and dropped to undetectable values 10 months in patient 1. No circulating antialveolar basement membrane antibodies were found by indirect IF in patient 2. Maintenance hemodialysis was required in both patients. Patient 1 died 1 year later without evidence of active Hodgkin's disease. The only common etiologic factor found in these two patients was a heavy inhalation of solvent vapors 15 days before the onset of anuria. This solvent contained sodium hydroxide, carboxymethylcellulose, nonylphenol, ethylene oxide and butane. It is suggested that: *(1)* solvent exposure may have resulted in these two patients in chemical damage to alveolar basement membrane, giving rise to anti-GBM antibodies and so initiating glomerulonephritis; *(2)* the possibility of solvent exposure should be carefully checked in each patient with apparent primary glomerulonephritis.

Regular dialysis treatment (RDT) in the elderly: A 5-year experience with 25 patients over 60-years-old. *B. Lebkiri, L. Boudier, A. Lemaire, N. K. Man, and P. Jungers. Department of Nephrology and INSERM U 25, Necker Hospital and Alma Dialysis Center, Paris, France.* Between January 1973 and June 1978, 25 patients (12 men, 13 women), aged from 60 to 71 years (mean, 65.6 years) were admitted to our RDT program, despite previous visceral or vascular lesions: 5 with coronary heart disease, 2 with cerebrovascular strokes, 4 with diabetes melitus. All were treated as center patients with a closed-batch dialysate delivery

system (Rhodial 75©) using isonatremic dialysate, and allowing precise control of ultrafiltration (UF). Twelve patients were dialyzed twice and 13 three times weekly. The mean interdialytic weight gain was 1.7 kg. Hypertension was present in 21 patients prior to RDT. Blood pressure was controlled by dialysis alone in 14, associated with drugs in 7. Twelve patients needed frequent blood transfusions (mean ± SD : 2.05 ± 0.75 packed cell units/patient/month) in order to avoid angina pectoris (7) or major asthenia (5) when hematocrit level fell below a critical value close to 30%. End-dialysis cardiac arrhythmias were prevented by adjusting dialysate potassium level in each patient, up to 4 mEq/liter, to prevent hypokalemia. Actuarial cumulative survival rate was 91.9, 82.6, and 71.5% at 1, 2 and 5 years, respectively, a mean mortality rate of 6% per year. Five deaths occurred: 2 from cerebro-vascular strokes, 1 from hepatic polycystic disease, 1 from gastrointestinal bleeding, and 1 from septicemia. Mean hospitalization rate was 11 days/patient/year. Rehabilitation was good in most cases. *Conclusion.* Results of RDT in patients over 60 can be satisfactory. Careful dialysis techniques including precise control of UF, prevention of end-dialysis hypokalemia, and correction of anemia may substantially reduce morbidity and mortality in this high-risk patient group.

Binding of tritiated 1-34 human parathyroid hormone to cellular membranes from rat kidney cortex. *N. Loreau, C. Lajotte, M. Lafaye, and R. Ardaillou. Hôpital Tenon, Paris, France.* Tritiated 1-34 human PTH (5 Ci/mmole) bound specifically to membranes purified from the rat renal cortex. Specificity of binding is based on the following: Binding equilibrium was reached both at increasing incubation times (10 min) and increasing PTH concentrations (2 μmole/liter); ^{3}H-PTH binding was inhibited by unlabeled hormone (approximately 100 nmole/liter for half inhibition of binding) and its analogues but neither by unrelated peptides nor inactivated PTH; addition of an excess of unlabeled PTH at equilibrium produced release of the tritiated hormone from its receptors; binding was linear with the concentration of membrane protein in the range 0.3 to 1.2 mg/ml; there was a close relationship between ^{3}H-PTH binding and adenylate cyclase stimulation by this tracer, both processes displaying similar K_D values (150 to 350 nmole/liter); the peptides which competed with ^{3}H-PTH for its binding sites also stimulated adenylate cyclase; trypsin treatment produced both a marked decrease in ^{3}H-PTH binding and suppression of the stimulation of adenylate cyclase. Nonspecific binding represented 15 to 30% of total binding. Binding was pH and temperature dependent, maximum binding being observed at pH 7.5 and 22° to 37° C. Binding also slightly increased with calcium concentration in the range of 0.5 to 5 mM. The degradation rate of ^{3}H-PTH was negligible during the time of the experiments and allowed study of binding at equilibrium without correcting hormone concentrations. These results demonstrate the presence of specific receptors for PTH linked to adenylate cyclase in the rat kidney.

Divergent acid-base disturbances in patients with advanced chronic renal failure (ARCF). *E. Matthys, N. Lameire, and S. Ringoir. University Hospital, Renal Division, De Pintelaan, Ghent, Belgium.* The incidence of tubular acidification disturbances in a nonselected group of patients (N = 20) with ACRF (creatinine clearance < 20 ml/min) was explored by association of a prolonged oral ammonium chloride (0.1 g/kg body wt) and the i.v. sodium bicarbonate loading test (1 ml/min of sodium bicarbonate, 6.72%). Urinary pH, ammonium (NH_4^+), titratable acid (TA) and bicarbonate (HCO_3^-) were determined and fractional and absolute HCO_3^- reabsorption were calculated over a wide range of plasma HCO_3^-. Net acid or base excretion were calculated at normal plasma HCO_3^- levels. A tubular acidification defect, defined as a $U_{pH} > 5.80$ after induction of systemic acidosis, was observed in 7/20 patients. In the 13 patients with adequate acidification, 2 subgroups could be differentiated: 7 with mean net acid excretion of 16.1 μEq/min at a mean plasma HCO_3^- of 24.5 mEq/liter; 6 with a mean net base excretion of 24.6 μEq/min at a mean plasma HCO_3^- of 25 mEq/liter. In the 7 patients with abnormally high urinary pH, 1 patient presented a net acid excretion of 6.65 μEq/min at a plasma HCO_3^- of 24 mEq/liter. The 6 others showed a pronounced HCO_3^- loss at normal P_{HCO_3} (mean net base excretion of 28.8 μEq/min). These 6 patients suffered from chronic interstitial nephritis. In the 7 patients with tubular acidification disturbances, the PTH levels were significantly higher than in the 13 others without acidification disturbances and with comparable degrees of renal failure. Calculation of the ratio of absolute HCO_3^- to sodium reabsorption showed no evidence of increased HCO_3^- reabsorption in acidotic patients with ARCF compared to patients with normal creatinine clearances (N = 12). It is concluded that the metabolic acidosis in ACRF can be divided into several different types of renal acidification disturbances. One third of these patients have additional tubular acidification problems, and approximately 50% of these patients present with impressive urinary loss of bicarbonate at normal plasma HCO_3^- concentrations.

Plasma catecholamine levels in hypertensive DOCA-salt rats. *M. de Mendonca, P. Guicheney and P. Meyer. Physiologie et Pharmacologie, INSERM U7 Hôpital Necker, Paris, France.* Plasma noradrenaline and adrenaline were measured in rats in experimental hypertension induced by administration of DOCA and a sodium-rich diet (DOCA-salt). A radioenzymatic assay of catecholamines was performed on blood samples obtained from conscious, unrestrained rats via a chronically implanted arterial catheter. It is shown that much lower concentrations are found under these conditions compared to those obtained after decapitation. The extreme sensitivity of plasma catecholamines in response to stimuli, such as the transfer of the rat from one room to another, is stressed. In DOCA-salt rats, noradrenaline was markedly raised after 5 weeks of treatment but did not greatly increase thereafter. This finding confirms previous works by others on decapitated or anesthetized animals. Plasma adrenaline was also increased showing the same type of evolution. On the other hand, the increase in blood pressure was gradual throughout the course of the 10-week observation period. Thus, the increase in blood pressure cannot be entirely accounted for by an overactivity of the catecholaminergic system. It was also shown that hemorrhage, even very moderate, induced a rise in plasma catecholamines and especially noradrenaline which is more marked in control than it is in DOCA-salt rats.

Role of renin in the enhancement of glycerol-induced ARF in the rat: Effects of SQ 14 225. *A. Mimran, E. Chevillotte, D. Casellas, and M. Dupont. Service of Medicine D, C.H.U. Montpellier, France.* Chronic sodium loading (HS) blunts and salt depletion (LS) enhances the severity of renal impairment in glycerol-induced ARF. This suggested an important role for the renin-angiotensin system in the pathophysiology of ARF. Glycerol (G) was injected into HS and LS rats and into animals treated by the orally active angiotensin converting enzyme inhibitor SQ 14 225 for 2 days prior to and during the 24-hour period following G (HS-SQ and LS-SQ groups). During the 24-hour period following G, urine volume did not change in HS and HS-SQ groups, and it decreased strikingly to 0.5 ± 0.4 and 1.3 ± 0.8 ml/24 hours in LS and LS-SQ groups, respectively. MAP was 108 ± 7 (HS), 107 + 9 (LS), and 84 ± 6 mm Hg (LS-SQ). Blood urea values (mg/100 ml) are shown in the table.

	No SQ	*SQ-treated*	*P*
HS	146 ± 28	102 ± 20	NS
LS	270 ± 10	332 ± 14	0.005
P	0.001	0.001	

These results do not suggest an important role for renin in the enhancement of ARF in LS rats. However, the lower MAP in LS-SQ rats and the possibility of a noninhibition of the intrarenal generation of angiotensin II might have prevented an improvement in renal function by SQ 14 225.

Hypokalemic myopathy with rhabdomyolysis and acute renal failure in the course of chronic licorice ingestion. *G. Mourad, P. Gallay, R. Oules, A. Mimran, C. Mion. Department of Nephrology, C.H.U., Nimes, France.* The occurrence of oliguric acute renal failure due to rhabdomyolysis in the course of chronic potassium depletion induced by long-term licorice ingestion has seldom been reported. A 51-year-old man, with hypertension of 6 month's duration, was admitted because of acute oliguria with symptoms of acute myopathy. This patient had ingested 5 g of licorice daily during the last 4 years. On admission, arterial pressure was 180/110 mm Hg; severe generalized weakness and oliguria were found. Blood urea was 140 mg/100 ml, plasma creatinine was 4.3 mg/100 ml, serum sodium and potassium were 146 and 1.4 mEq/liter, serum chloride was 69 mEq/liter; decompensated metabolic alkalosis was found: pH, 7.6; bicarbonate, 51 mEq/liter; P_{CO_2}, 54 and P_{O_2} 45 mm Hg. Hematest was positive in the urine. In addition serum CPK was 9264 mU/ml (normal, 16 to 35), and LDH was 1840 mU/ml (normal, 110 to 160). Supine PRA was 0.32 ng/ml/hr (normal 1.3 to 3.0), and plasma aldosterone concentration was 3.6 ng/100ml (normal, 4 to 12). Peritoneal dialysis treatment was undertaken, and urine output reappeared within 3 weeks. Complete recovery of renal function was subsequently observed. Muscle biopsy showed evidence of macrovacuolar degeneration and myolysis. Renal biopsy, performed on the day 12, showed extensive tubular lesions and intratubular casts. Among the factors proposed to explain this syndrome, the role of chronic potassium depletion by licorice, associated with furosemide ingestion and muscular exercise 2 days before anuria, is possible. The finding of low-renin aldosterone hypertension is compatible with an important role of licorice.

Study of tubular basement membrane deposits after human renal transplantation. *C. Orfila, J. Rakotoarivony, D. Durand, and J. M. Suc. INSERM U-133, C.H.U. Rangueil, Toulouse, France.* A study of tubular basement membrane of 65 allografted kidneys by immunofluorescence shows IgG and/or C3 deposits in 27 cases. Tubular lesions were studied by light and electron microscopy. The pattern of immune deposits was linear in 6 cases (with presence of anti-TBM antibodies by RIA and/or indirect IF in 4 cases), granular in 16 cases, and atypical with linear and granular segments in 5 cases. In 7 cases, IF study of the patient's own kidneys before transplantation revealed no deposits. Electron microscopy study of renal tubules showed that the alterations found in the TBM were thickening, lamellation, and/or deposits along TBM. The prognostic significance of these deposits was assessed. The presence of linear deposits did not indicate poor prognosis. In contrast, the detection of granular deposits along the TBM indicated poor graft outcome.

Nephropathies induced in rabbits by antibodies directed against renal basement membranes. *J. Rakotoarivony, C. Orfila, P. Bardos, P. Mahieu, J. P. Muh, and J. M. Suc. Groupe de recherche INSERM U 133 and Laboratoire d'Optique Electronique du C.N.R.S. Toulouse, France.* Heterologous antitubular basement membrane antiserum was produced in a sheep by injecting highly purified rabbit tubular basement membrane (TBM). This serum was injected into rabbits and its effects compared to those of a heterologous antiserum directed against rabbit glomerular basement membrane (GBM) produced in the same conditions. Both antisera induce a severe proliferative glomerulonephritis. However, the anti-TBM antiserum produces, in addition, an immune tubulopathy characterized by the linear fixation of autologous IgG along the TBM, by the presence of a strong, albeit focal, tubular atrophy and by a significantly higher glycosuria incidence. It is suggested that the antibodies directed against antigens shared by both GBM and TBM polypeptides are able to induce a severe glomerulonephritis in rabbits, whereas an immune tubular disease is induced by the antibodies directed against antigens present in TBM polypeptides only.

Improved technique for removal of formalin and other toxic elements from hemodialyzers. *P. Ramperez, G. Claret, G. Deschodt, C. Emond, C. Granoleras, C. Mion, H. Mion, and S. Shaldon. Dept. of Nephrology and Dept. of Biochemistry, University Hospital, Nimes/Montpellier, France.* Since the observation of Ogden (CFDT 1973) that subclinical administration of formalin to patients occurred regularly when a standard 2-liter saline rinse of formalin-sterilized dialyzers was used and the occasional report of ethylene oxide toxicity, little progress has been made to improve rinsing techniques. We have developed an economic and simple method to eliminate trace toxic elements from dialyzers. The AMICON hemofilter (Tm30) was shown to be capable of removing pyrogen, bacteria, and particulate matter and produce fluid suitable for i.v. injection at a rate of 250 ml/min using a standard blood pump. A test for filter intactness using Sephadex blue was developed and used before each test. The filter was placed between the arterial blood line and a Dylade DS generator designed to supply dialysate rinsing fluid to the blood compartment of the dialyzer. Twelve hollow-fiber dialyzers (Travenol, 1.5 m^2) were sterilized in 4% formalin and then rinsed with 2 liters of saline. They were resterilized and then rinsed for 30 min on the Amicon/DS (with 7 liters of sterile fluid). After use, the sterilization with formalin was repeated and the rinse-out technique reapplied before the second use. Formalin in the venous effluent was measured by the Hantzsch method, detecting less than 1 μg/ml of formalin. With standard rinse technique, the effluent contained 30.0 $\pm$ 6.81 μg/ml (1 SD) (although negative to clinitest and Schift). With the Amicon technique, the effluent contained 7.8 $\pm$ 0.7 μg/ml before first use and 8.5 $\pm$ 1.61 μg/ml after reuse. Three patients have used the same Amicon filter (resterilized in formalin after each use) three times per week for 6 months to rinse their dialyzers and also to restitute the blood after dialysis. This technique permits a more efficient removal of trace toxic elements such as formalin and will possibly eliminate the problem of anti-N like antibody formation. At the same time, it elminates the need for routine i.v. sterile fluids for priming and restituting blood from the dialyzer.

Purine metabolism and urolithiasis. *R. J. Réveillaud, M. Daudon, M. F. Protat, G. Ayrole, and V. Vinther-Harder. Centre hospitalier de Saint-Cloud, France.* In man, purine urolithiasis is nearly always uric or uratic, but the rare xanthic and adeninic forms should be thoroughly examined. The analysis of 277 urinary stones (excluding recurrences of the same type) gives 16.2% of uric lithiasis, pure and mixed (with 5.4% of pure forms and 10.8% of mixed forms); ammonium urate has been found in 6.3% of cases, always under the mixed forms with 2, 3, or 4 components; the two forms of uric acid and ammonium urate comprise 22.5% of the 277 analyzed lithiases. Furthermore, the presence of oxalate has been observed in 66.7% of the uric lithiasis and in 30% of the uratic lithiasis, excluding uric acid. The different chemical and crystalline forms are: anhydrous uric acid, dihydrated uric acid, ammonium urate, rarely sodium urate, xanthine, hypoxanthine, and 2,8-dihydroxyadenine. Among present methods of chemical analysis, only infrared spectrography can precisely distinguish all these different types. In particular, it allows the diagnosis of rare forms of 2,8-dihydroxyadenine, which has always been, up to now, confused with uric lithiasis; in effect this purine reacts, as uric acid and urate, with murexide and phosphotungstic acid, which are widely used for stone analysis. Our experiments after surgery have shown a new case of this type, by I. R. spectrography. A total deficit of erythrocyte adenine phosphoribosyl transferase (APRTase) was found and confirmed that urolithiasis occurs only in homozygote forms. Although rare, this disease has deleterious effects through relapses and by leading to renal insufficiency. Treatment with allopurinol has been started under strict supervision because the consequences of the double enzyme deficiency in APRTase and in xanthine oxydase, are, as yet, unknown.

Recurrent hemolytic-uremic syndrome: Cortical necrosis with irreversible renal failure in course of oral contraceptive therapy. *G. Rifle, J. M. Chalopin, R. Genin, E. Justrabo, J. Severac, and F. Bulte. Unité de Néphrologie, Hôpital du Bocage, Dijon, France.* Five recurrences of hemolytic-uremic syndrome (HUS) occurred in a 17-year-old woman. The disease had started when she was 3-years-old after upper respiratory infection. Three recurrences occurred between the age of 3 and 8 after measles, varicella, then new upper respiratory infection. These four episodes resolved without any specific treatment. At the age of 17, 14 days after bacterial angina, a new typical episode occurred with irreversible renal failure due to bilateral cortical necrosis. At that time she had been taking oral contraceptives for 4 months. Renal biopsy showed lesions of thrombotic microangiopathy; immunofluorescence was negative. Immunologic investigations performed since the beginning of the last episode and followup during the first 12 months of hemodialysis showed the following results: there was a permanent decrease of serum C3 level and an intermittent decrease of factor B. No C3NeF activity was found. Search for circulating immune complexes by the C1q binding method was persistently negative. Cryoglobulinemia was found on several occasions. This case can be added to the 35 registered HUS with recurrent episodes. Various etiologic factors before each episode is a good argument for the uniqueness of this syndrome. Immunologic studies suggest the possible activation of C3 without circulating immune complexes or without C3NeF activity.

Results obtained with an ultrafiltration control monitor (UFcM) in patients treated by maintenance hemodialysis (MH). *J. Rottembourg, D. Jacq, J. P. Durande, P. Rojas. Service de Néphrologie, Groupe Hospitalier Pitié-Salpétrière, Paris, France.* An ultrafiltration control monitor (UFcM) allows a programmed and visualized withdrawal of fluid in patients treated by hemodialysis. With the equipment used in this study, a definite amount of dialysis fluid is poured at regular intervals into a closed circuit. The volume of ultrafiltrate (UF) withdrawn from the patient is collected in a glass tube. The efficiency of the UFcM has been studied in four patients during two consecutive periods, each of 4 months' duration. During the first period, two patients were treated with single-pass hemodialysis and with dialyzers fitted with cuprophane or cellophane membranes. Two patients were dialyzed with artificial kidneys fitted with polyacrylonitrile membranes (PAN) and with a closed-circuit dialysis system. During the second period, the four patients were treated with the same types of dialyzers and with the UFcM. Pre- and post-dialysis plasma levels of urea, creatinine, potassium, and phosphorus were similar during the two periods. In the two patients treated with PAN membranes, the mean extraction ratios of urea creatinine and phosphorus ($N = 9$) determined 15 min after the start of dialysis are similar with both technique. Clinical incidents (cramps, hypotensive episodes) were less frequent during the use of the UFcM. The visualization of UF, and its permanent control during dialysis, allows a better quality of care delivered to patients treated by maintenance hemodialysis distributed by HOSPAL.

Drug-induced liver cytolysis in hemodialyzed patients. *P. Simon and A. Meyrier. Centre Hospitalier, Saint-Brieuc, and Hôpital Tenon, Paris, Methods.* Fifty-three patients who had been dialyzed for 10 to 132 months (mean, 35 ± 22) were followed from Jan 75 to Aug 78, for 7 to 44 months (mean, 24.5 ± 12.9 mo). In 20, one to four liver biopsies allowed *(1)* classification of liver pathology (chronic persistent hepatitis (CPH), chronic aggressive hepatitis (CAH), or cirrhosis (C)). *(2)* HBsAg detection by immunofluorescence and *(3)* measurement of liver iron content by the method of Barry. Followup of transaminase level revealed 67 cytolytic episodes (SGPT > 40 IU); for each episode, a drug enquiry was made. *Results. Group 1:* 21/27 chronic HBsAg carriers exhibited 49 cytolytic episodes, 15 of which occurred at the time of HBsAg positivation. Thirty others coincided with drug administration: i.v. polymaltose iron (10 times), allopurinol (7), alpha-methyldopa (3), fluoxymesterone (2), aspirin (1), phenylindane-dione (1), oxomemazine-paracetamol (1), lincomycin (1), indometacin (1), I.N.H. (1) ampicillin (1), oxazepam (1). Twenty-seven liver biopsies taken 8 to 45 months after the beginning of chronic serum HBs antigenemia disclosed CPH in 14, CAH in 12, C in 1, iron overload in 23, and HBsAg in 23. *Group 2:* Twelve out of twenty-six HBs-negative patients (comprising 8/13 HBs antibody carriers) exhibited 18 cytolytic episodes. Twelve coincided with drug administration: i.v. polymaltose iron (3 times), allopurinol (4), aspirin-paracetamol (2), amidopyrin (1), indometacin (1), quinine (1). Five liver biopsies taken in 4 cases disclosed CPH in 3, a normal liver in 2, hepatic iron overload in 5. HBsAg was negative in all samples. *Conclusions. (1)* Except for the initial period of HBsAg positivation, the majority of cytolytic episodes coincided with drug administration. *(2)* Any aggravation of hepatitis in a HBs + dialysis patient must lead to immediate withdrawal of all nonindispensable drugs. *(3)* Some "non A, non B" hepatitis in hemodialysis patients are drug related.

Excretion and synthesis of basement membrane disaccharide units in Masugi nephritis. *M. Sternberg, P. de Grandpré, V. Pelletier, and M. Carter. Groupe de biochimie et d'immunopathologie, Département de Biochimie, Faculté de Médecine, Université Laval, Québec, Canada.* During nephrotoxic nephritis in the rat, an increased urinary excretion of glycosylgalactosyl hydroxylysine and of galactosyl-hydroxylysine has been observed in the autologous phase of the disease. This is mainly due to an elevation of the polypeptide-bound fraction of these hydroxylysyl glucosides with a molecular weight over 1,000 daltons. The levels of both urinary hydroxylysyl glucosides were correlated with proteinuria. Their increased excretion appears to originate in the lysed glomerular basement membrane. At the same stage of nephrotoxic nephritis an increased glycosyl transferase activity could be demonstrated in the isolated glomeruli, correlated with albuminuria, attesting a higher turn-over of the disaccharide units of the glomerular basement membrane.

Adult hemolytic uremic syndrome successfully treated by plasma exchange. *P. Vialtel, F. Chenais, E. Dechelette, F. Bayle, P. Couderc, and D. Cordonnier. Centre Hospitalier Régional et Universitaire de Grenoble, Grenoble, Cedex, France.* A 37-year-old Algerian male was hospitalized 4 days after the beginning of abdominal pain and fever. He was being treated for asthma with steroids, salbutamol, and desensitization. On admission, physical examination and laboratory tests revealed acute renal failure (diuresis, 200 ml/24 hr; serum creatinine, 123 mg/liter; proteinuria, +++), hemolytic anemia, and numerous fragmented red cells. The platelet count was 16,000/mm^3. There was neither bleeding nor other symptoms of intravascular coagulation. A kidney biopsy performed 13 days after admission confirmed the hemolytic uremic syndrome without extrarenal involvement (skin, muscle, and gingival biopsies). Screening for bacterial, viral, or parasitic infection was negative. No cryoglobulin or immune complexes were found; complement system was normal. Fibrin degradation products were absent from serum and urine. Test for falciformation of red cells and hemoglobin electrophoresis were normal. Treatment consisted of plasma exchange (Haemonetics Model 30) at day H1, H2, H4, and H7, hemodialysis at H1 and H4. Heparin was started at H3 and continued for 30 days. At H2, after the second plasma exchange, the platelet count reached 135,000. At H4, there were no more fragmented cells, diuresis was 2 liters. At H32, serum creatinine was 9 mg/liter. Nine months after this episode, the patient is doing well, and no recurrence has occurred. The reason for such a result in a usually very severe disease is not known. It is not explained by removal of circulating immune complexes or fibrin degradation products. Removal of a pathogenic factor is hypothetical. As was suggested by recent works, the beneficial effect of massive infusion of fresh plasma must be considered.